Optic Neuropathy

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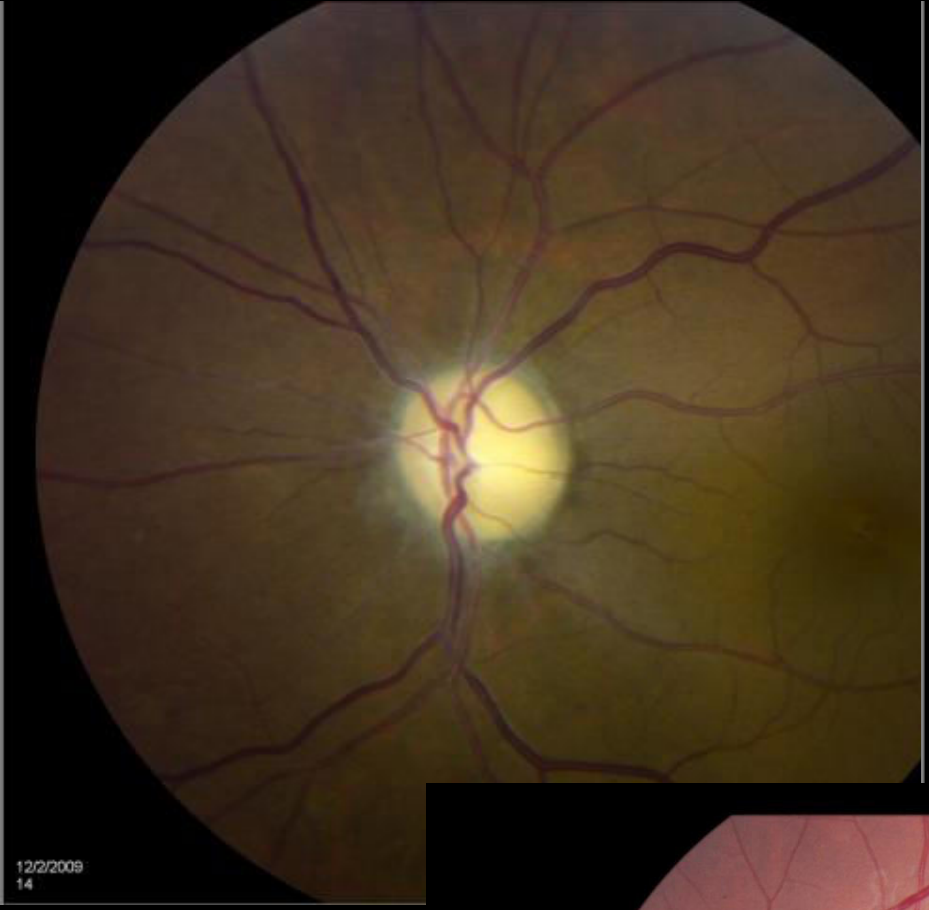
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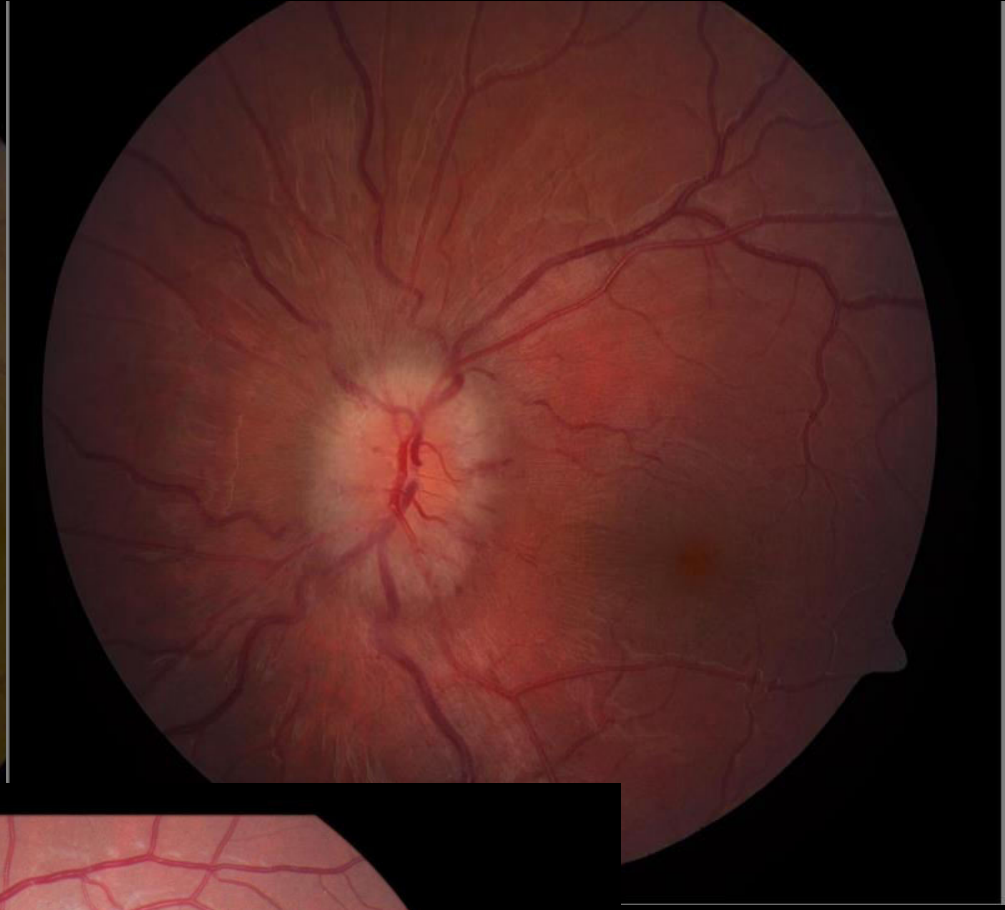
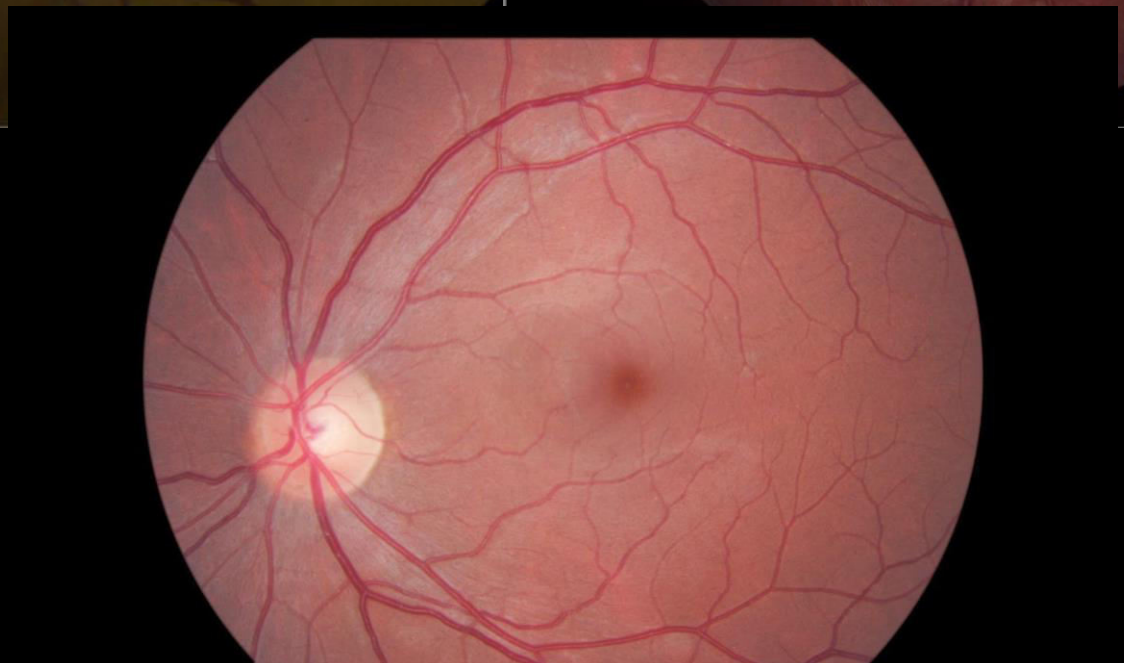
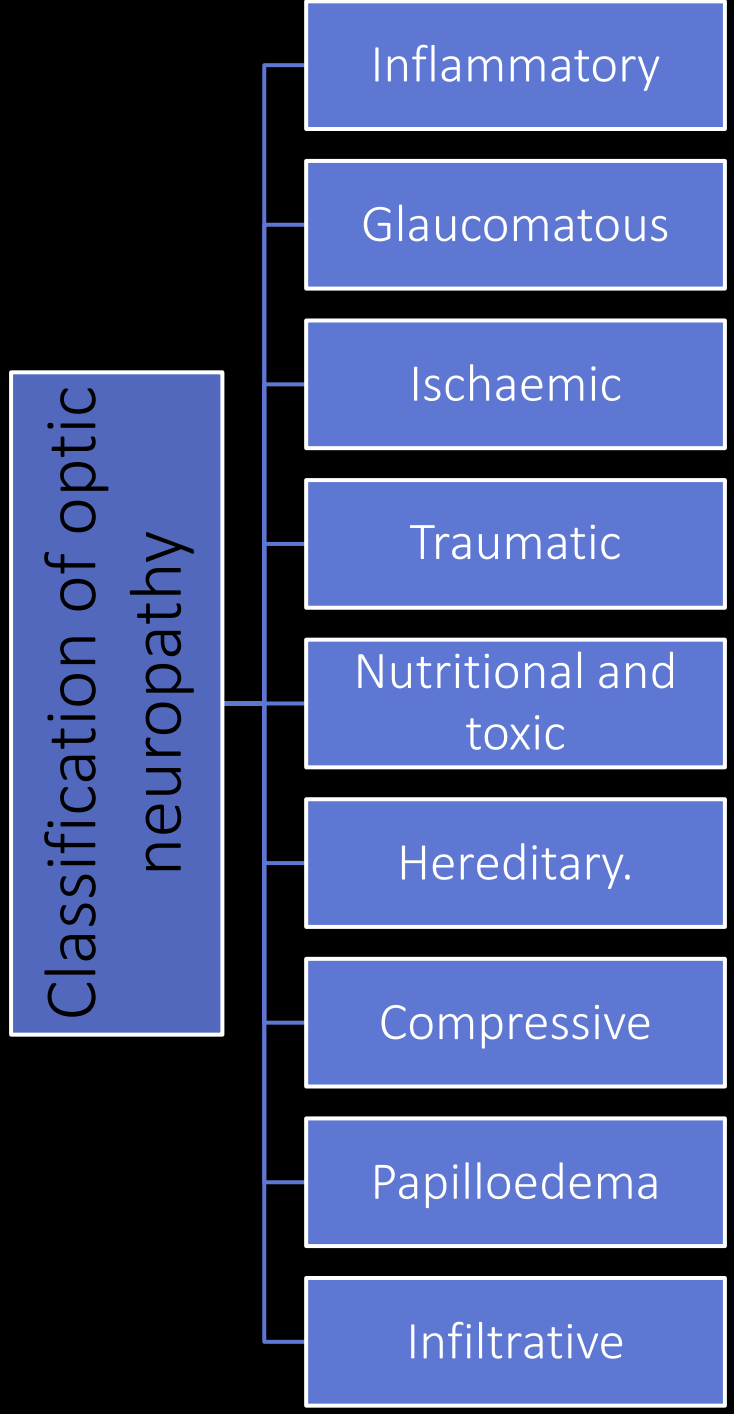




Optic neuropathy

Optic neuropathy is damage to the optic nerve from any cause.
Damage and death of these nerve cells, or neurons, leads to characteristic features of optic neuropathy.


Classification of optic neuropathy
Inflammatory
Glaucomatous
Ischaemic
Traumatic
Nutritional and toxic
Hereditary.
Compressive
Papilloedema
Infiltrative

Optic Neuropathies : Causes

- **Demyelinating**
- Inflammatory
- Non-arteritic Ischemic
- Arteritic Ischemic
- Traumatic

Rapid onset

- Infiltrative
- Compressive
- Hereditary
- Radiation
- Paraneoplastic
- Toxic/nutritional

Gradual onset

Hereditary Optic Neuropathy

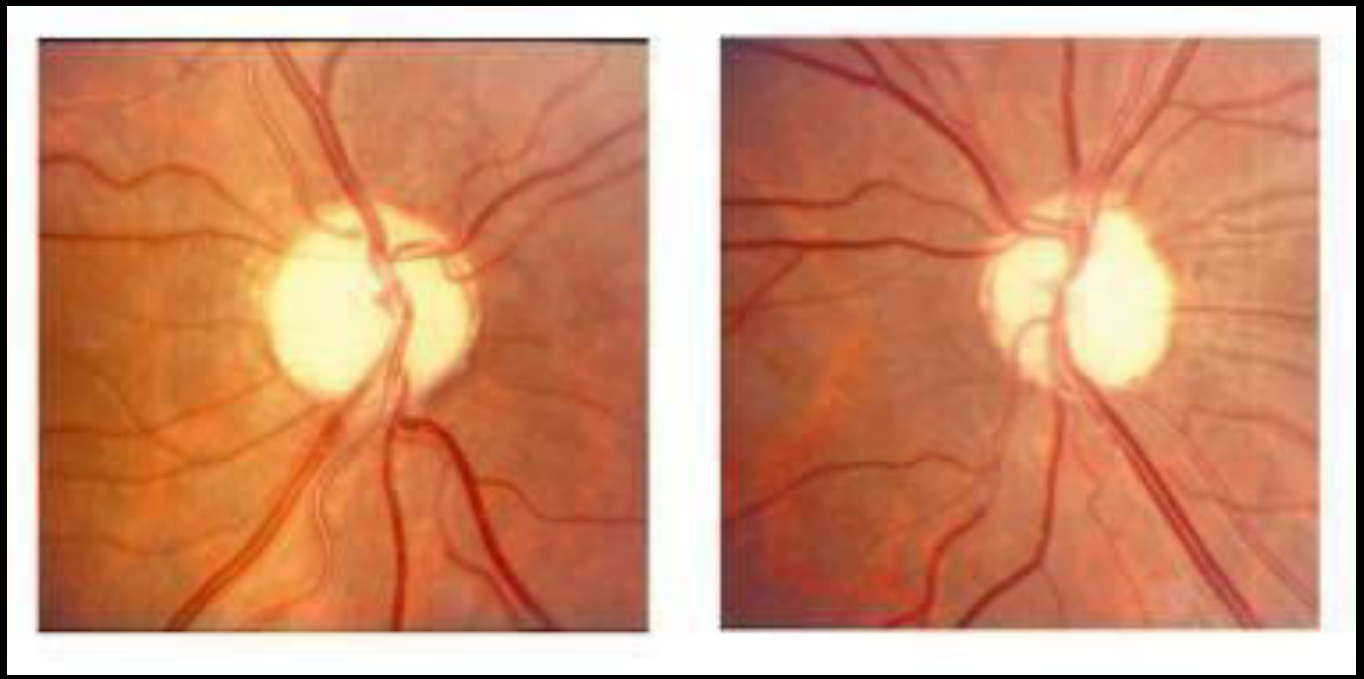
- AD
- AR
- XL

HEREDITARY OPTIC NEUROPATHY: AD (KJERS' TYPE)

- 1st decade of life
- Bilateral symmetric visual loss.
- Bilateral central or cecocentral scotomas.
- Color vision deficit .

HEREDITARY OPTIC NEUROPATHY: AD (KJERS' TYPE)

- The optic disc : temporal pallor and in some cases severe excavation and cupping.



HEREDITARY OPTIC NEUROPATHY: BEHR SYNDROME

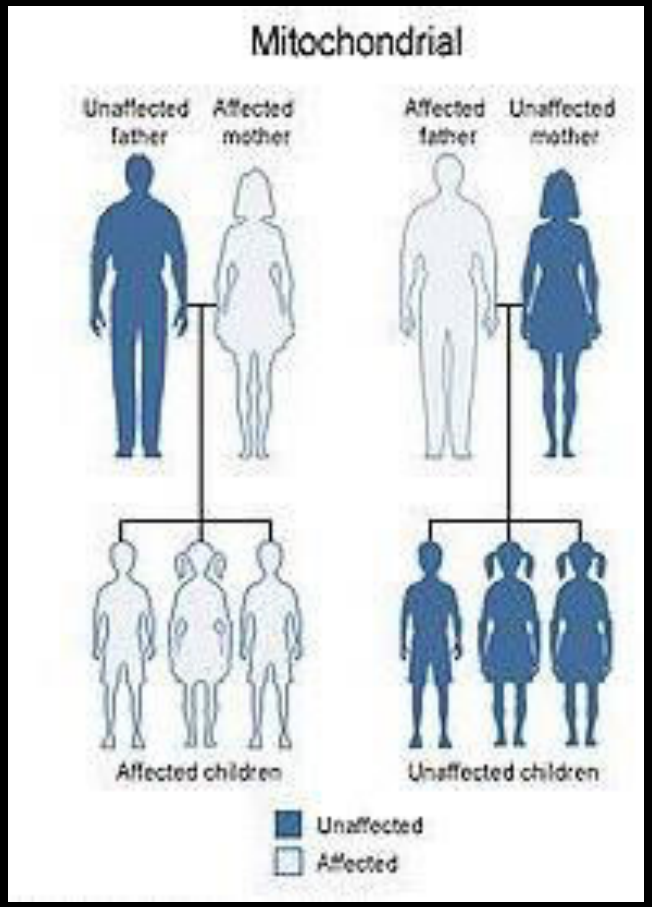
- Inheritance is AR
- Presentation is in early childhood with reduced vision.
- Optic atrophy is diffuse.
- Prognosis is variable, with moderate to severe visual loss and nystagmus.
- Systemic abnormalities include spastic gait, ataxia and mental handicap.

HEREDITARY OPTIC NEUROPATHY; WOLFRAM SYNDROME

- also referred to as DIDMOAD (diabetes insipidus, diabetes mellitus, optic atrophy and deafness).
- 1st year of life
- inheritance being AR, AD or via the maternal mitochondrial line.
- Optic atrophy is diffuse and severe and may be associated with disc cupping.

HEREDITARY OPTIC NEUROPATHY: LHON

- leber hereditary optic neuropathy
- LHON has 4 primary mitochondrial mutations
- M>F
- The frequencies of mutation may vary across different countries



HEREDITARY OPTIC NEUROPATHY: LHON

- Acute unilateral, painless, visual loss.
- some cases may stay asymptomatic or have a chronic course
- Sequential bilateral involvement may occur weeks or months later.

May demonstrate neurological as peripheral neuropathy, ataxia, Dystonia and cardiac conduction defects

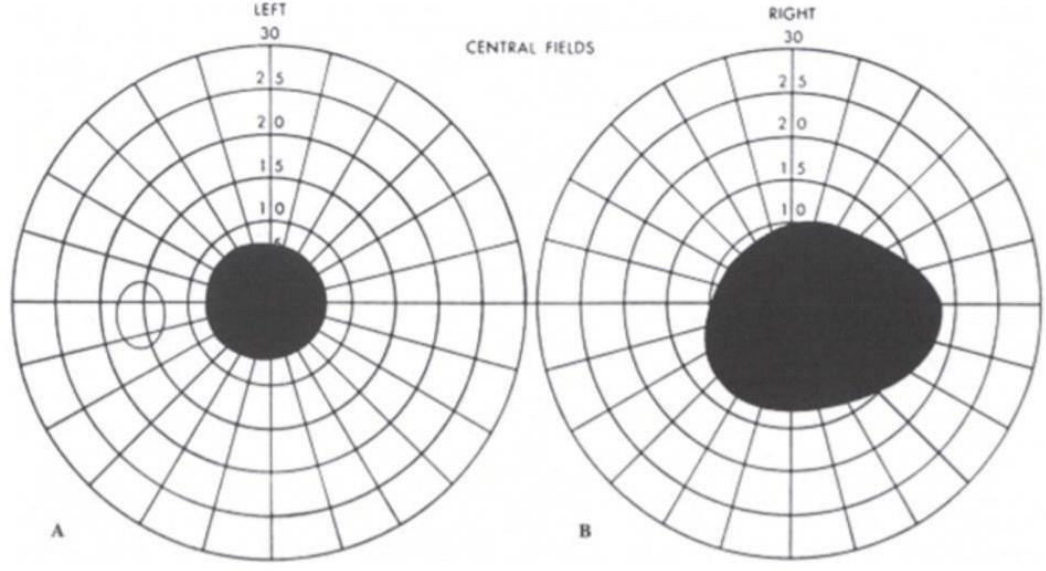
HEREDITARY OPTIC NEUROPATHY: LHON

- Occasionally, optic nerve pallor can be seen initially. Because of the wide age range (6–80 years old) at which LHON may present, it is frequently misdiagnosed

- Young patients are often diagnosed as optic neuritis and older patients as ischemic or infiltrative optic neuropathy.

HEREDITARY OPTIC NEUROPATHY: LHON

- Visual filed defects tend to be central or cecocentral as the papillo-macular bundle is first and most severely



HEREDITARY OPTIC NEUROPATHY: LHON

- Fundoscopy may show disk swelling, thickening of the peripapillary retinal nerve fiber layer

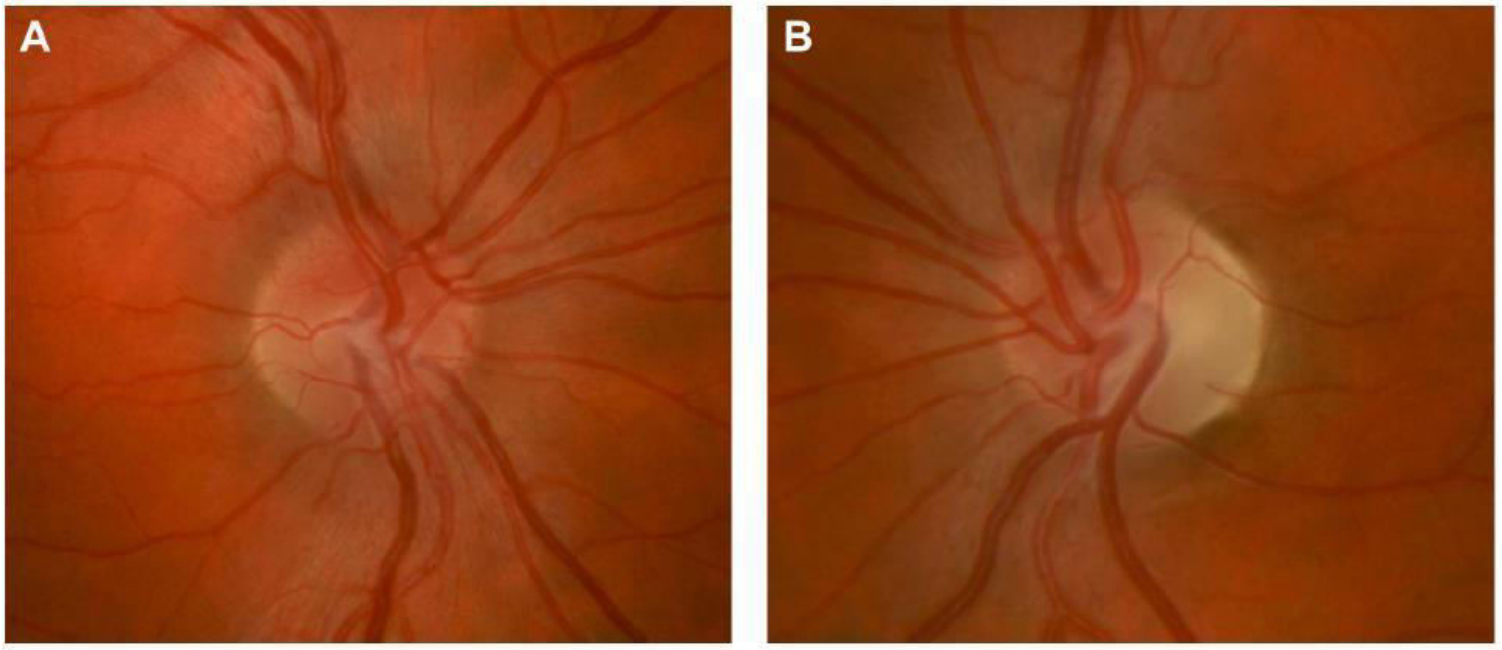


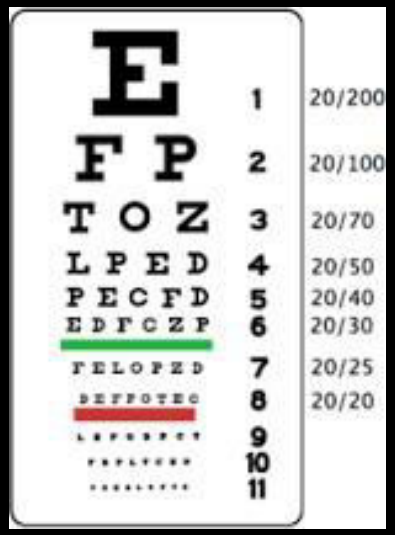
Figure 2 Right optic nerve (**A**) of a patient with acute LHON-related vision loss showing mild hyperemia, blurring of the disc margin, and elevation of the optic nerve head from swelling of the peripapillary retinal nerve fiber layer. LHON-related vision loss in the left eye had occurred 6 months prior leading to prominent temporal optic nerve pallor (**B**) from atrophy of the retinal nerve fiber layer.
Abbreviation: LHON, Leber hereditary optic neuropathy.

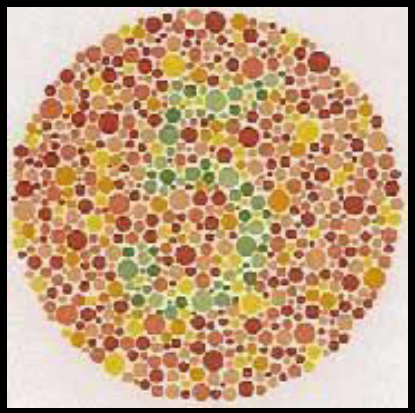
TREATMENT •

- Apart from symptomatic measures such as low vision aids, treatment is generally ineffective.
- Dietary deficiencies should be avoided, particularly of B12.
- Smoking and excessive alcohol consumption should be discouraged, theoretically in order to minimize mitochondrial stress.
- Idebenone appears to be neuroprotective in this condition and may have a role to play in some patients.
- Gene therapy is under active investigation

Toxic Optic Neuropathies

- Visual acuity may vary from minimal reduction to no light perception (NLP) in rare cases.
- Most patients have 20/200 vision or better.
- Color vision should be assessed because dyschromatopsia is a constant feature in these conditions.



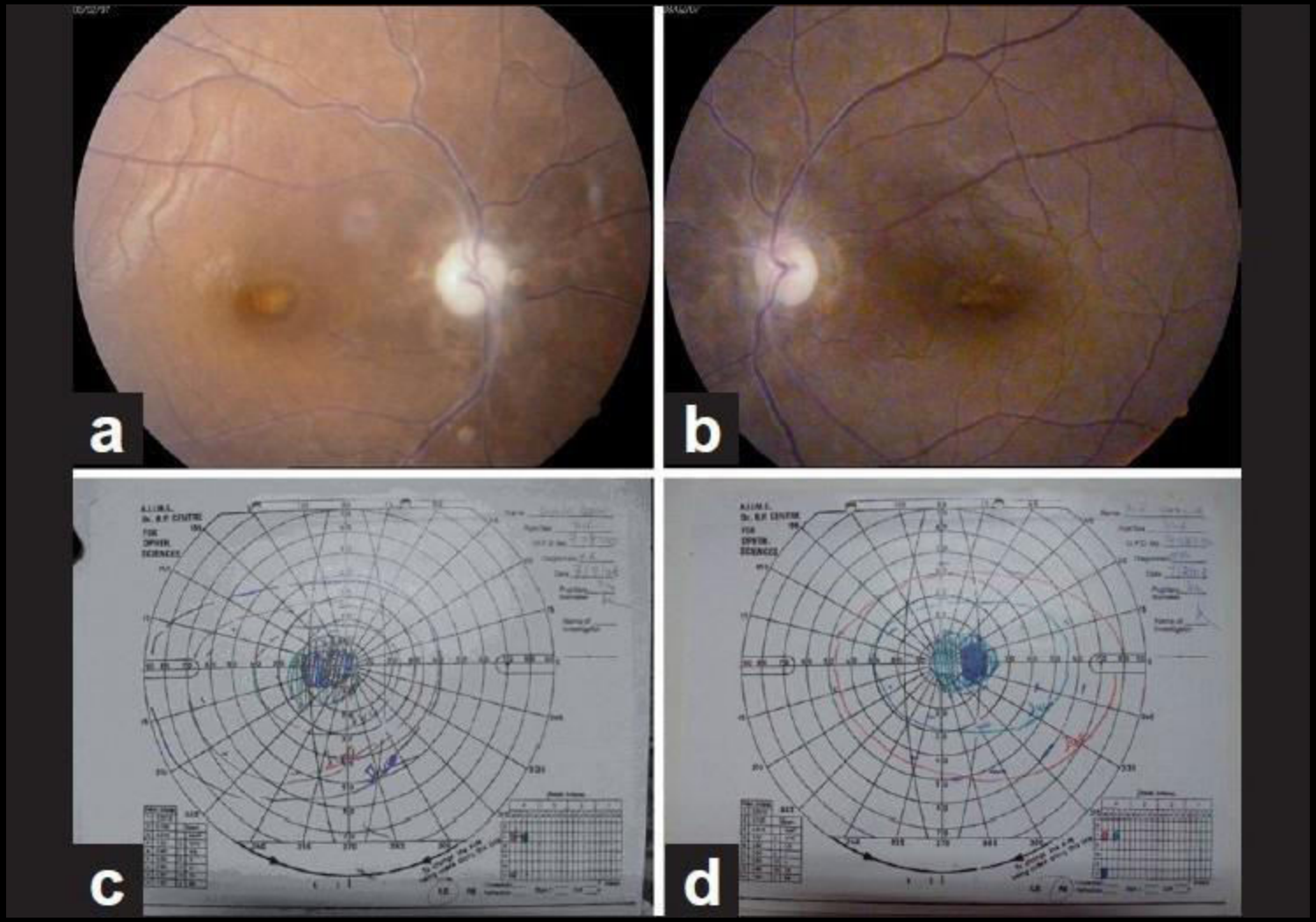


Toxic Optic Neuropathies

- In the early stages of toxic optic neuropathies, most patients also have normal-appearing optic nerves, but disc edema and hyperemia may be seen in some intoxications, especially in acute poisonings.

- Papillomacular bundle loss and optic atrophy develop after a variable interval depending on the responsible toxin.

TOXIC OPTIC NEUROPATHIES



TOXIC OPTIC NEUROPATHIES

Alcohols: Methanol, ethylene glycol (antifreeze)

Antibiotics: Chloramphenicol, sulfonamides, linezolid

Antimalarials: Chloroquine, quinine

Antitubercular drugs: Isoniazid, ethambutol, streptomycin

Antiarrhythmic agents: Digitalis, amiodarone

Anticancer agents: Vincristine, methotrexate

Heavy metals: Lead, mercury, thallium

Others: Carbon monoxide, tobacco

Nutritional Optic Neuropathies

THE CLINICAL PRESENTATION AND BASIC PATHOPHYSIOLOGY ARE SIMILAR TO TON

Most often, they present as a non-specific retrobulbar optic neuropathy.

Currently, the treatment is limited to the intensive use of vitamins with variable results in individual cases, and to the implementation of preventive measures, when feasible.

Nutritional Optic Neuropathies

- Optic disc may be normal or slightly hyperemic in the early stages.
- In a small group of patients with hyperemic discs, small splinter hemorrhages on or off the disc.
- Several months to years later , papillomacular bundle dropout and temporal disc pallor, followed by optic atrophy.

Nutritional Optic Neuropathies

- Thiamine (vitamin B1)
- Cyanocobalamin (vitamin B12)
- Pyridoxine (vitamin B6)
- Niacin (vitamin B3)
- Riboflavin (vitamin B2)
- Folic acid

Nutritional Optic Neuropathies

TOBACCO ALCOHOL AMBYLOPIA (TAA)

- TAA is an old term for a constellation of elements that can lead to a mitochondrial optic neuropathy.

- The classic patient is a man with a history of heavy alcohol and tobacco consumption.

Nutritional Optic Neuropathies

TOBACCO ALCOHOL AMBYLOPIA (TAA)

- Combined nutritional mitochondrial impairment, from vitamin deficiencies (folate and B-12) classically seen in alcoholics, with tobacco- derived products, such as cyanide

- It has been suggested that the additive effect of the cyanide toxicity,and deficiencies of thiamine, riboflavin, pyridoxine, and b12 result in TAA

Toxic Optic Neuropathies: Other agents

- Hypovitaminosis A – night blindness (nyctalopia), keratomalacia.

- Hypervitaminosis A – yellow skin and conjunctiva, pseudotumor cerebri (papilledema), retinal hemorrhage.

TREATMENT

- Treatment Consideration should be given to co-management with a general physician or neurologist
- Dietary revision with formal nutritional advice, incorporating increased fruit and leafy green vegetable intake.
- Abstention from alcohol and tobacco is a priority
- Vitamins. A daily multivitamin preparation, plus thiamine (100 mg twice daily) and folate (1 mg daily). • Intramuscular hydroxocobalamin (vitamin B12) injections.
- Exposure to the identified agent should be discontinued immediately in cases due to medication or environmental toxicity

THANK YOU